



# UNITED STATES PATENT AND TRADEMARK OFFICE

UNITED STATES DEPARTMENT OF COMMERCE  
United States Patent and Trademark Office  
Address: COMMISSIONER FOR PATENTS  
P.O. Box 1450  
Alexandria, Virginia 22313-1450  
www.uspto.gov

APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
10/544,146	05/05/2006	Shyam S. Mohapatra	USF-T193XC1	9945
23557 7590 11/03/2010 SALIWANCHIK LLOYD & SALIWANCHIK A PROFESSIONAL ASSOCIATION PO Box 142950 GAINESVILLE, FL 32614				
EXAMINER SCHNIZER, RICHARD A				
ART UNIT		PAPER NUMBER		
1635				
NOTIFICATION DATE		DELIVERY MODE		
11/03/2010		ELECTRONIC		

**Please find below and/or attached an Office communication concerning this application or proceeding.**

The time period for reply, if any, is set in the attached communication.

Notice of the Office communication was sent electronically on above-indicated "Notification Date" to the following e-mail address(es):

euspto@slspatents.com

### Office Action Summary

**Application No.**

10/544,146

**Applicant(s)**

MOHAPATRA ET AL.

**Examiner**

Richard Schnizer

**Art Unit**

1635

**Period for Reply** -- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

**Status**

- 1) ☒ Responsive to communication(s) filed on 24 February 2010.
- 2a) ☐ This action is **FINAL**. 2b) ☒ This action is non-final.
- 3) ☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

**Disposition of Claims**

- 4) ☒ Claim(s) 42, 52, 56, 57, 59, 64-67, 69, 71-75 and 77-83 is/are pending in the application.
- 4a) Of the above claim(s) \_\_\_\_\_ is/are withdrawn from consideration.
- 5) ☐ Claim(s) \_\_\_\_\_ is/are allowed.
- 6) ☒ Claim(s) 42, 55, 57, 59, 64-67, 69, 71-75, 77, and 79-83 is/are rejected.
- 7) ☒ Claim(s) 56 and 78 is/are objected to.
- 8) ☐ Claim(s) \_\_\_\_\_ are subject to restriction and/or election requirement.

**Application Papers**

- 9) ☐ The specification is objected to by the Examiner.
- 10) ☐ The drawing(s) filed on \_\_\_\_\_ is/are: a) ☐ accepted or b) ☐ objected to by the Examiner.  
Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).  
Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).
- 11) ☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

**Priority under 35 U.S.C. § 119**

- 12) ☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).
- a) ☐ All b) ☐ Some \* c) ☐ None of:
1. ☐ Certified copies of the priority documents have been received.
  2. ☐ Certified copies of the priority documents have been received in Application No. \_\_\_\_\_.
  3. ☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

\* See the attached detailed Office action for a list of the certified copies not received.

**Attachment(s)**

- 1) ☐ Notice of References Cited (PTO-892)
- 2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-940)
- 3) ☒ Information Disclosure Statement(s) (PTO/SB/08)  
Paper No(s)/Mail Date 7/19/2010
- 4) ☐ Interview Summary (PTO-413)  
Paper No(s)/Mail Date \_\_\_\_\_
- 5) ☐ Notice of Informal Patent Application
- 6) ☐ Other: \_\_\_\_\_

## **DETAILED ACTION**

### ***Continued Examination Under 37 CFR 1.114***

A request for continued examination under 37 CFR 1.114, including the fee set forth in 37 CFR 1.17(e), was filed in this application after final rejection. Since this application is eligible for continued examination under 37 CFR 1.114, and the fee set forth in 37 CFR 1.17(e) has been timely paid, the finality of the previous Office action has been withdrawn pursuant to 37 CFR 1.114. Applicant's submission filed on 7/19/10 has been entered.

Claims 53-55, 58, 61, 62, 70, and 76 were canceled and claims 82 and 83 were added.

Claims 42, 52, 56, 57, 59, 64-67, 69, 71-75, and 77-83 are pending and under consideration.

### ***Priority***

Applicant's claim for the benefit of a prior-filed application under 35 U.S.C. 119(e) or under 35 U.S.C. 120, 121, or 365(c) is acknowledged. Applicant has not complied with one or more conditions for receiving the benefit of an earlier filing date under 35 U.S.C. 119(e) as follows: The later-filed application must be an application for a patent for an invention which is also disclosed in the prior application (the parent or original nonprovisional application or provisional application). The disclosure of the invention in the parent application and in the later-filed application must be sufficient to comply with

the requirements of the first paragraph of 35 U.S.C. 112. See *Transco Products, Inc. v. Performance Contracting, Inc.*, 38 F.3d 551, 32 USPQ2d 1077 (Fed. Cir. 1994).

This Application claims priority to provisional applications 60/320108 (filed 4/15/2003), and 60/319964 (filed 2/21/2003). All instant claims are directed to a method using "a vector comprising at least one gene suppressing cassette". In view of the specification as filed, e.g. Figures 1B and 1C, this phrase must be interpreted as embracing vectors comprising at least 2 or 3 gene suppressing cassettes. The claims require each cassette to comprise a polynucleotide encoding an siRNA wherein the siRNA is linked to a promoter. The 60/320108 application does not clearly provide support for vectors comprising more than one gene suppressing cassette wherein each cassette comprises a promoter and siRNA. The '108 specification discusses vector construction at paragraph 14. This passage appears to indicate that two oligonucleotides, each encoding a pol III transcription terminator (TTTTT), were separately cloned into separate U6 expression vectors, one as an ApaI/XhoI fragment, and the other as an XhoI/EcoRI fragment. There is no clear support for inserting each of two promoter/siRNA cassettes into a single vector. Accordingly, the '108 application does not provide support for the instant claims under 35 USC 12, first paragraph.

All instant claims read on delivery of siRNA expression cassettes to dendritic cells (DC), polynucleotides comprising SEQ ID NOS: 3 or 4, and target sequences common to 4 serotypes of Dengue virus (DV). Instant claim 57 requires a chitosan conjugate. Instant claim 42 is drawn broadly to viral vectors, and instant claim 43 has a specific embodiment limited to an adenoviral vector. Instant claims 72-75, 77-79, 82,

and 83 require administration to DC in vivo. The 60/319964 application does not support any of these embodiments. Because all instant claims read on delivery of siRNA expression cassettes to dendritic cells (DC), polynucleotides comprising SEQ ID NOS: 3 or 4, and target genes common to 4 serotypes of Dengue virus (DV), the '964 application is not considered to fully support the claims under 35 USC 112, first paragraph.

Accordingly the effective filing date of the instant claims is considered to be that of PCT/US2004/005566, i.e. 2/23/2004.

### ***Claim Rejections - 35 USC § 103***

The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

(a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negated by the manner in which the invention was made.

Claims 42, 52, 59, 64, 69, 71-73, 75, 77, and 80-82 are rejected under 35 U.S.C. 103(a) as being unpatentable over Iversen et al (US 20050096291), Raviprakash et al (J. Virol. 69(1):69-74, 1995), Adelman et al (J. Virol. 76(24): 12925-12933, 2002), Tuschl et al (US 7,056,704), and Yu et al (PNAS 99(9): 6047-6052, 2002).

Iversen taught methods of inhibiting Dengue virus replication in a human by administration of an antisense oligonucleotide directed against the Dengue genome. SEQ ID NO: 27 inhibited replication of four different Dengue serotypes in Vero cells. See and paragraph 180. See abstract and paragraphs 34, 36, 40, 50, 153, 154, 158,

Example 3, Figs 5A-D, and claim 16. Iversen taught several routes of delivery including intravenous, subcutaneous, transdermal, and topical to the skin. See paragraph 154.

Raviprakash taught a method of inhibiting expression of DV gene products in mammalian LLCMK/2 cells by microinjection of antisense directed at the 5' end of the portion of the RNA encoding the structural proteins, and the 3' end of the virus genome. Cells were exposed to DV after delivery of antisense. The target regions were 15 bases in length. One oligonucleotide was directed to a target sequence common to all four DV serotypes. Two oligonucleotides were directed to a non-structural protein (ns5a). See abstract; paragraph bridging pages 69 and 70; first full paragraph on page 70; Fig. 1 on page 70; page 73, column 2, lines 22-26.

Adelman taught a plasmid vector encoding a inverted repeat siRNA directed against Dengue virus structural gene prM RNA, and its use to inhibit DV infection by administration to mosquito cells in vitro. See abstract.

Yu taught expression cassettes encoding hairpin siRNAs and their use in mammalian cells. See abstract. Yu disclosed the concepts of including the cassettes in nonviral vectors and in viral vectors. See last paragraph on page 6052.

Tuschl stated that "siRNAs are extraordinarily powerful reagents for mediating gene silencing" and that "siRNAs are effective at concentrations that are several orders of magnitude below the concentrations applied in conventional antisense or ribozyme gene targeting experiments." See column 23, lines 15-20.

It was clear to those of ordinary skill in the art at the time of the invention that there was interest in inhibiting DV replication in human cells, particularly in view of the

Art Unit: 1635

teachings of Iversen and Raviprakash, and that RNA interference represented an alternative to antisense methods of gene suppression. Therefore, it would have been obvious to one of ordinary skill in the art at the time of the invention to modify the method of Iversen by substituting an expression vector encoding an shRNA directed against DV for the antisense oligonucleotide of Iversen. One would have been motivated to do so in order to obtain the advantages disclosed by Tuschl. One of ordinary skill appreciates that shRNAs operate through the same pathway as siRNAs, and would reasonably expect shRNAs to deliver performance superior to that obtained using antisense in view of the teachings of Tuschl. One would have had a reasonable expectation of success because the technology for expressing shRNAs in mammalian cells was available prior to the time of the invention (see Yu above), and there was no reason to doubt that it would have functioned in vivo in human cells.

It would have been similarly obvious to target any of the DV genomic RNA regions set forth by Iversen, Raviprakash, or Adelman because the genome of DV is a positive strand RNA encoding a single polyprotein, such that cleavage of any of the targets would have been expected to inhibit DV replication, particularly those at the 5' genome end, e.g. those directed against prM. One would have been motivated to include expression cassettes for more than one siRNA in order to increase the level of inhibition achieved.

With regard to whether or not the siRNA vector is administered prior to or after DV infection, Iversen taught that treatment could be either prophylactic or post-infection (paragraph 71). In any case, an infected individual will produce infectious virus such

that treatment of an infected individual would be considered to result in the treatment of cells that had not yet become infected, but would become infected subsequent to treatment.

Absent evidence to the contrary, delivery to dendritic cells is considered to be inherent in intravenous administration.

Claims 57 and 83 are rejected under 35 U.S.C. 103(a) as being unpatentable over Iversen et al (US 20050096291), Raviprakash et al (J. Virol. 69(1):69-74, 1995), Adelman et al (J. Virol. 76(24): 12925-12933, 2002), Tuschl et al (US 7,056,704), and Yu et al (PNAS 99(9): 6047-6052, 2002) as applied to claims 42, 52, 59, 64, 69, 71-73, 75, 77, and 80-82 above, and further in view of Yu et al (US 6852528).

The teachings of Iversen, Raviprakash, Adelman, Tuschl, and Yu (2002) are summarized above. These references can be combined to render obvious methods of inhibiting DV replication in a human by intravenous administration of a vector encoding an shRNA directed against DV RNA.

These references do not teach a vector conjugated with chitosan. However, one of ordinary skill appreciates that there is a wide variety of gene delivery techniques which one may employ interchangeably as a matter of design choice. Among these are microinjection (the method used by Raviprakash), lipofection (used by Adelman (2002) and Yu (2002)). Yu ('528) also taught that a variety of methods could be used to deliver nucleic acids to cells including microparticle formation with polycations such as chitosan-based compounds, as well as liposome-mediated transfection and



microinjection. See column 22, lines 17-44; column 23, lines 30-47; and column 31, lines 22-38. It would have been obvious to one of ordinary skill in the art to select any of these commonly used transfection techniques, as they were all well recognized in the art as exchangeable alternatives.

Claims 65-67, 74 and 79 are rejected under 35 U.S.C. 103(a) as being unpatentable over Iversen et al (US 20050096291), Raviprakash et al (J. Virol. 69(1):69-74, 1995), Adelman et al (J. Virol. 76(24): 12925-12933, 2002), Tuschl et al (US 7,056,704), and Yu et al (PNAS 99(9): 6047-6052, 2002) as applied to claims 42, 52, 59, 64, 69, 71-73, 75, 77, and 80-82 above, and further in view of Hope et al (US Patent 6,136,597)

The teachings of Iversen, Raviprakash, Adelman, Tuschl, and Yu (2002) are summarized above. These references can be combined to render obvious methods of inhibiting DV replication in a human by intravenous administration of a vector encoding an shRNA directed against DV RNA.

The cited references did not teach adeno-associated virus vectors.

Hope taught that expression cassettes could be delivered by a variety of viral or non-viral vectors, including plasmid, adeno associated virus, adenoviral, retroviral, lentiviral, polioviral and herpes viral vectors. See column 13, line 16, to column 14, line 23. MPEP 2144.06 indicates that when it is recognized in the art that elements of an invention can be substituted, one for the other, while retaining essential function, such elements are art-recognized equivalents. An express suggestion to substitute one

equivalent component or process for another is not necessary to render such substitution obvious. Furthermore, MPEP 2144.07 indicates that the selection of a known material based on its suitability for its intended use supports the determination of prima facie obviousness. The various vectors described by Hope are all considered to be equivalent platforms for carrying expression cassettes, so it would have been obvious to use any of them to deliver the expression cassette described above. The structural characteristics of the AAV vector of Hope are indistinguishable from those recited in the instant claims, so the functional characteristic of not causing acute inflammation in dendritic cells is considered to be inherent.

### ***Response to Arguments***

Applicant's arguments filed 7/19/10 have been fully considered but they are not persuasive.

Applicant asserts that the rejections all rely on the Iversen patent publication, and that the instant claims are entitled to an earlier filing date than is the Iversen publication. This is unpersuasive for the reasons set forth above under "Priority".

Applicant asserts that the claimed invention is non-obvious because the combined references did not provide one of ordinary skill in the art at the time of the invention with a reasonable expectation of successfully inhibiting the expression of Dengue virus genes within a human host suffering from Dengue virus infection. Applicant relies for support on Subramanya et al (2010, of record) who indicated that a hurdle for RNAi therapeutics is the specific delivery of small interfering RNA (siRNA) to

relevant cell types, and that development of a method to introduce siRNA into DCs would be an important step toward using RNAi therapeutically to suppress viral replication in Dengue infection. Applicant asserts that the important step of developing a method to introduce Dengue virus-targeted siRNA into DCs to inhibit viral replication was first achieved by the instant inventors, pointing to Examples 8 and 9 of the instant specification which show that adeno associated virus vectors can transduce DCs, and encoded siRNAs can inhibit subsequent DV infection and DV-induced apoptosis in DCs. Applicant further asserts that none of the cited references shows nucleic acid delivery to DCs, and concludes that the combined references did not provide one of ordinary skill in the art at the time of the invention with a reasonable expectation of success.

With regard to claims 42, 52, 57, 59, 64-67, 69, 71, and 80, Applicant's arguments are unpersuasive because they are based on the issue of the predictability of transfecting DCs, but do not address the predictability of transfecting other DV targets, such as hepatocytes and macrophages. Note that only claims 72 and dependents explicitly require transfection of DCs. None of the other claims requires transfection of DC cells. An et al (Virology 23: 70-77, 1999, of record) provided evidence that DV could infect hepatocytes in vivo and replicate therein (see abstract and discussion). Applicant has presented no evidence or argument that one of ordinary skill at the time of the invention would not have reasonably expected to be able to deliver siRNA to DV-infected hepatocytes in vivo. Applicant has indicated previously that DV infects and replicates in macrophages (Applicant's Remarks filed 10/27/08 at page 5, penultimate sentence of second full paragraph), and this position is supported

by Subramanya (see abstract). However, Applicant has presented no evidence or argument that one of skill in the art at the time of the invention could not have accomplished delivery to macrophages *in vivo* with a reasonable expectation of success using the methods of the combined references.

Applicant's arguments with specific regard to the unpredictable nature of transfecting DCs are also unpersuasive. While, Subramanya indicated that non-selective transfection methods may not work well for primary hematopoietic cells, this does not necessarily mean that one of ordinary skill would not have had a reasonable expectation of successfully combining the cited references to arrive at the claimed invention. To circumvent the potential problem of non-selective transfection methods not working well for DCs, Subramanya developed a vector that targeted DCs through the use of a peptide shown to bind DCs specifically. This does not mean that those of ordinary skill in the art at the time of the invention believed that DCs could not be transfected in the absence of such a vector. Applicant has presented no clear evidence that one of ordinary skill would have doubted that at least some DCs would have been transfected by the methods of the combined references, and that DV gene expression would have been inhibited in those transfected cells. On the other hand, the prior art showed that DCs could be transfected *in vivo*. Palmowski et al (*J. Immunol.* 172: 1582-1587, 2004) demonstrated that dendritic cells could be directly transfected *in vivo* by intravenous administration of lentiviral vectors. See also the following references which disclose *in vivo* transfection of DCs: Condon et al (*Nature Medicine* 2(10): 1122-1128, 1996), Song et al (*Proc. Nat. Acad. Sci. USA* 94: 1943-1948, 1997), Porgador et al (*J.*

Exp. Med. 188(6): 1075-1082, 1998), Barratt-Boyes (J. Immunol. 164: 2487-2495, 2000), and Larregina et al (Gene Therapy 8: 608-617, 2001).

Applicant asserts that the Raviprakash reference, alone or in combination with the other references, would not have provided one of ordinary skill with a reasonable expectation of success. Applicant asserts that Raviprakash used monkey kidney cells, not dendritic cells, and disclosed that unmodified antisense oligonucleotides were not effective in bringing about significant inhibition of DV. With regard to the cells used by Raviprakash, the Office notes that there is substantial evidence of record that dendritic cells were known to be transfectable in vivo (see the previous paragraph). Regarding the effectiveness of antisense in inhibiting DV, Fig. 2, panel A on page 71 of Raviprakash shows that unmodified antisense oligonucleotides were ineffective, but standard phosphorothioate oligonucleotides showed greater inhibition, and C-5 propyne-modified antisense oligonucleotides showed very significant inhibition (see panels B-D). These antisense technologies were well known in 1995 when Raviprakash was published, and so it is reasonable to conclude that Tuschl (with an earliest claimed priority date of 12/1/2000) was aware of them when indicating that siRNA inhibition was superior to antisense inhibition. Moreover, there is no reason of record to expect that antisense or siRNA approaches in general would not function in DCs. Accordingly one of ordinary skill would have had a reasonable expectation of improving on the results of Raviprakash when substituting siRNA for antisense, and when delivering to DCs.

Applicant asserts that the oligonucleotide of Raviprakash that target a sequence conserved in all 4 DV serotypes (denoted as "3'a") showed limited efficacy and that in contrast the instant specification exemplified very effective inhibition of DV infection and DV-induced apoptosis in human dendritic cells. In response it is noted that methods limited to the specific siRNAs used in the experiments relied on for this argument have been indicated as allowable.

Applicant asserts that the Adelman reference is not relevant to inhibiting DV in human cells or a human. By way of clarification, the Adelman reference is relevant to the rejection because it disclosed the concept of RNA interference inhibition of DV replication, showed that it could function in cells, and disclosed accessible target regions for RNAi.

Applicant asserts that it is important to consider whether or not blockade of host molecules such as TNF-alpha also interferes with a possible antiviral effect that might outweigh its pathogenic potential, and relies for support on Subramanya (2010). Applicant asserts that the instant specification shows that interfering RNA did not induce acute inflammation in human DC as determined by the level of pro-inflammatory cytokines including TNF-alpha. The relevance of this argument is unclear to the Examiner. Applicant appears to be arguing limitations that are not in the claims. It is noted that claim 74 requires that the adenoviral vector does not induce inflammation in dendritic cells, but this is considered to be an inherent property of adenoviral vectors. The claims do not recite any limitations regarding blockade of TNF alpha.

The remainder of Applicant's response addresses rejections based on those discussed above, wherein the rejections cite further references. Applicant argues that these further references do not remedy perceived deficiencies in the previous references. These arguments are unpersuasive for the reasons set forth above.

In conclusion, the Office finds that one of ordinary skill in the art at the time of the invention would have had a reasonable expectation of achieving some level of DV inhibition in humans through the combination of the cited references. This is particularly true in view of the fact that antisense had been shown to inhibit DV replication in vitro, and RNA interference was considered to be superior to antisense for purposes of gene expression inhibition. Iversen suggested delivery of nucleic acids to the skin, and the prior art of record demonstrated that DCs could be transfected by this route. Moreover, it was known that DV could infect hepatocytes in vivo, and that nucleic acids could be delivered to liver cells by intravenous administration. Accordingly one of ordinary skill would have had a reasonable expectation of obtaining some level of DV inhibition through the in vivo administration of siRNAs directed to DV RNA.

For these reasons the rejections are maintained.

### ***Conclusion***

No claim is allowed.

Claims 56 and 78 are objected to because they depend from rejected claims, but would be allowable if rewritten in independent form with all of the limitations of the claims from which they depend.

Any inquiry concerning this communication or earlier communications from the examiner(s) should be directed to Richard Schnizer, whose telephone number is 571-272-0762. The examiner can normally be reached Monday through Friday between the hours of 6:00 AM and 3:30. The examiner is off on alternate Fridays, but is sometimes in the office anyway.

If attempts to reach the examiner by telephone are unsuccessful, the Examiner's supervisor, Christopher Low, can be reached at (571) 272-0951. The official central fax number is 571-273-8300. Any inquiry of a general nature or relating to the status of this application or proceeding should be directed to (571) 272-0547.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see <http://pair-direct.uspto.gov>. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free). If you would like assistance from a USPTO Customer Service Representative or access to the automated information system, call 800-786-9199 (IN USA OR CANADA) or 571-272-1000.

/Richard Schnizer/  
Primary Examiner, Art Unit 1635



**Search Notes (continued)****Application/Control No.**

10/544,146

**Applicant(s)/Patent under  
Reexamination**

MOHAPATRA ET AL.

**Examiner**

Richard Schnizer

**Art Unit**

1635

**SEARCHED**

Class	Subclass	Date	Examiner

**INTERFERENCE SEARCHED**

Class	Subclass	Date	Examiner

**SEARCH NOTES  
(INCLUDING SEARCH STRATEGY)**

	DATE	EXMR
updated	10/24/2010	RS